ORAL FLECAINIDE FOR PREVENTION OF PAROXISMAL ATRIAL FIBRILLATION: PREDICTIVE VALUE OF THE RESPONSE TO TRANSESOPHAGEAL ATRIAL STIMULATION.

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We studied 22 pts,mean age 48 years,who had multiple episode of primary paroxismal atrial fibrillation (PAF). Transesophageal atrial stimulation (TAS) was performed after all conventional antiarrhythmic drugs had been stopped for at least 5 half-lives before testing,using a moderately aggressive protocol: single and double extrastimuli during sinus rhythm and during 600 and 400 msec cycle lenght pacing and 8" atrial bursts at incremental rate from 180 to 300 bpm.All pts had inducible,sustained (a 1 min duration) PAF at the time of basal TAS.All pts received oral Flecainide (F) 150 mg bid (pts 60 Kg,100 mg bid). All pts underwent a second TAS after at least 10 days of oral F therapy. Fourteen pts were considered responders (sustained PAF no more inducible) and 8 non responders (sustained PAF still inducible).There was no difference between responders and non responders in regard to age,sex, left atrial dimension,duration of PAF,electrophysiologic parameters before and after F and plasma level of F.All pts were discharged on chronic oral F.During a follow-up of 12±4 months,2 responders (14%) and 5 non responders(63 %) had recurrences of PAF (P 0.005).

We conclude that oral F is effective and safe for treating pts with PAF. TAS is a rapid,simple and not expensive method in predicting the response to oral F.

SUMMARY OF A MULTICENTER TRIAL OF RECAINAM FOR SUSTAINED VENTRICULAR ARRHYTHMIAS.

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A multicenter trial was conducted to evaluate the efficacy of recainam (R), a 1C antiarrhythmic agent, in 80 pts with inducible sustained ventricular tachycardia (VT-S) or ventricular fibrillation (VF). Sixty pts (75%) presented with VT-S, 19 (24%) with VF, and 1 pt with syncope. These pts failed 311 prior antiarrhythmic drug trials (mean=4±2, range 1-11).

IV Recainam: Of 56 pts undergoing electrophysiologic study (EPS) on IV R, 5 (9%) had no inducible VT-S or VF. IV R increased VT-S cycle length (CL) in 39 pts (76%) and increased CL by more than 100 msec in 3 pts (16%). There was no difference in VT-S CL between the clinical VT-S, VT-S at control EPS, and VT-S on IV R (328±53 ms vs. 310±57 ms vs. 326±62 ms, respectively, p=ns).

Oral Recainam: Of 74 pts receiving oral R, 14 pts (19%) developed spontaneous VT-S or VF before repeat EPS. One pt died during loading and one was withdrawn for recurrent nonsustained VT. Therefore, of 58 pts undergoing EPS on oral R, 4 (7%) pts had no VT-S or VF inducible. VT-S CL increased in 38 pts (72%) and by more than 100 msec in 8 pts (15%). Oral R increased VT-S CL compared to control EPS (386±89 vs. 311±56, p=.001).

Adverse Effects: Besides spontaneous VT-S, no serious arrhythmias or conduction disturbances were reported. No serious hematologic disturbances occurred and other side effects were minor and reversible.

Conclusions: In pts with drug refractory VT-S or VF: 1) IV or oral R makes 8% of pts noninducible. 2) R increases VT-S CL by >100 msec in 15% of pts. 3) 19% of pts had spontaneous VT-S or VF during oral R loading.

FLECAINIDE INCREASES ENERGY FOR ATRIAL FIBRILLATION CARDIOVERSION

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Flecainide (Flec) may increase energy requirements for ventricular fibrillation cardioversion. Since Flec is used for atrial fibrillation (AF), we examined whether Flec alters the energy necessary for cardioverting AF. We cardioverted (apex-scapula patches) 37 patients (pts) with AF using successive shocks of 50 joules (J), 100 J, 200 J and 360 J. The distribution, by J, for cardioversion for Flec versus non-Flec pts was:

	50J	100J	200J	360J
Flec (n=14)	0/14	1/14	6/14	7/14
*non-Flec (n=23)	4/23	10/23	7/23	2/23

(Median joules for cardioversion: Flec = 300J; non-Flec = 100J; *P<.001; Wilcoxon-rank sum). Other antiarrhythmic drugs (Ia, III) distributed randomly among energies. Flec and non-Flec pts did not differ in ejection fraction (39±10% vs 41± 15), age (62±9 yrs. vs 65±10) left atrial size (3.9±1 cm vs 4.1±1) or disease. Three pts crossed from non-Flec to Flec with at least 1 energy level increase. Pauses greater than 4 sec were seen in 3/14 Flec pts and 0/23 non-Flec pts (*P=.05, chi sq). These data suggest that Flec increases energies for AF cardioversion and may produce pauses after the shock.

ANTIARRHYTHMIC SUPPRESSION OF SUPRAVENTRICULAR ARRHYTHMIA: IS EMPIRICAL THERAPY A SAFE APPROACH IN THE PRESENCE OF HEART DISEASE?

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Twelve patients (Group I) with either coronary artery disease (8 Pts) or cardiomyopathy (4 Pts), with mean left ventricular ejection fraction of 39.2% and supraventricular arrhythmias treated with Class IA or C antiarrhythmic agents (AA) were compared with a control group of 10 Pts with normal heart and supraventricular arrhythmias on similar AA regimen (Group II). No Pt had any documented ventricular arrhythmia prior to AA therapy. Programmed ventricular stimulation (PVS) was performed in all Pts (Groups I and II) while on AA controlling supraventricular arrhythmias. Eight of Group I Pts had inducible monomorphic ventricular tachycardia (IMVT) while none of Group II had inducible ventricular tachycardia. Subsequent PVS off of AA was performed on these 8 Group I Pts and no IMVT was documented.

Conclusion: In Pts with structural heart disease and left ventricular dysfunction, empirical AA therapy for suppression of supraventricular arrhythmias could produce a reentrant substrate for potentially malignant ventricular arrhythmias. Thus PVS guided AA treatment may be a safer approach.